

The mechanics of urethral closure, incontinence and midurethral sling repair. Part 2 further experimental validation (1993-2003)

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In: Part 1, The original 1990 science behind the MUS, the hypothesized closure mechanisms, and the prototype MUS itself were presented. The next phase of MUS development began in 1990 in collaboration with the late Ulf Ulmsten. It had two arms

1. Further development of the prototype MUS.
2. Further anatomical, imaging, urodynamic studies to validate the role of PUL in the closure mechanisms.

A second series of prototype MUS operations performed under LA/sedation resulted in a permanently implanted polypropylene sling and the MUS as is known today. The tape was elevated until no urine leaked on coughing. This demonstrated that the artificial PUL neoligament needed to be at a specific length to work. Anatomical, EMG and video ultrasound, and X-ray studies confirmed three directional muscles contracted pubourethral (PUL) and uterosacral (USL) ligaments. The contribution of the horseshoe shaped rhabdosphincter (RS) to continence was directly tested with pressure measurements under live surgery conditions. It was concluded that the RS was responsible for pressure generation but not continence. Continence was a consequence of intraurethral resistance to flow created by the distal and proximal urethral closure mechanisms, both governed ultimately by the Law of Poiseuille.

Conclusions: The key element in curing USI is creation of a competent PUL using the collagenous neoligament surgical principle described in Part 1. This creates a firm insertion point for the three directional muscle forces, restoring their contractile strength and closure.

KEYWORDS

anatomy, arc of Gilvernet, midurethral sling, neoligament, pubourethral ligament, pubovesical ligament, stress incontinence

Dr. Alan Wein led the peer-review process as the Associate Editor responsible for the paper.

The 1st author PP is the co-inventor of the MUS, Integral Theory and TFS. BA has made major scientific contributions to the colorectal aspects of the Integral Theory and has acted as a teacher for the MUS.

Ethics Where relevant, EC approval was obtained for studies at the time.

1 | INTRODUCTION

Part 1 described some of the original scientific studies which led to the midurethral sling (MUS). Observations discovered while developing the prototype midurethral sling operations in the late 1980s could not be explained by the pressure transmission theory. They could be explained by the Integral Theory of Female Urinary Incontinence, which stated¹

1. Urethral closure was activated by reflex contraction of three directional striated muscle forces, pubococcygeus (PCM), levator plate (LP), and longitudinal muscle of the anus (LMA). These contracted against the pubourethral (PUL) anteriorly and the cardinal/uterosacral ligament complex posteriorly. Separate from this was a 4th muscle, puborectalis (PRM) which was hypothesized as the muscle harnessed (by squeezing upwards) to cutoff the urinary stream and for Kegel exercises.
2. Loose ligaments, a consequence of birth, or age related collagen/elastin damage, weakened the contractile force of the closure muscles, resulting in USI, and other bladder dysfunctions. Furthermore, anatomical support of the loose ligament with a sling restored anatomy and continence.

To understand how the MUS works first requires an understanding of normal urethral closure, how a loose PUL causes USI, and the mechanisms by which the MUS operation restores continence. The main aim of part 2 is to explore the scientific basis of these mechanisms.

With reference to Figure S1: vector forces act forwards and backwards against the pubourethral ligament “PUL” (arrows); a downward force acts against the uterosacral ligament “USL” to angulate LP downwards; this action rotates the bladder downwards around the pre-cervical arc of Gilvernet “arc” to close off the urethra at bladder neck, much like kinking a hose. (see Videos 3 & 4).

1.1 | Proof that the directional movements are caused by muscle actions

EMG electrodes placed in the anterior vagina and posterior fornix confirmed muscle activity in the front and back pelvic muscles during coughing and straining, in accordance with the organ movements seen in Figure S1. The directional movements, Figure S1 and the EMG readings, Figure S2, are not possible with pressure transmission hypotheses.

1.2 | Anatomical validation of muscles and ligaments hypothesized to close the urethra, Figure 3–5

A series of cadaveric and live anatomical studies were made^{2–5} to confirm the position of the hypothesized closure muscles in

Figure S1. With reference to Figure S3, m. pubococcygeus “PCM” is consistent with the forward vector, levator plate “LP” with the backward vector and conjoint longitudinal muscle of the anus “LMA,” with the downward vector. Puborectalis muscle “PRM” lies below LP and can lift all the organs forward as is seen during squeezing (Kegel movement). Note the insertion point of PUL into PCM. Figure S4 more precisely defines how PUL separates the two parts of pubococcygeus muscle; the anterior part of PCM closes the distal urethra; the lateral part of PCM sweeps backwards to help form the levator plate “LP.” Figure S5 is a live anatomical demonstration of the two parts of PUL, inserting into PCM and the middle part of the urethra,⁵ (not the upper 2/3 as described by Zacharin⁶). Note also the external urethral ligament “EUL” which attaches the urethral orifice to the anterior surface of the pubic bone. Biopsies taken at the time demonstrate the presence of collagen, nerves, smooth muscle, and blood vessels. This indicates that the pubourethral ligaments are active contractile structures. Figure S6 schematically illustrates how laxity in the distal urethral closure mechanism functions.

1.3 | How a loose PUL causes USI

Figure S6 shows how a weak PUL elongates with effort. At the same time, the forward and backward forces acting against it weaken because their insertion point is loose. The urethra goes from closed mode “C” to open mode “O.” Unilateral pressure with a hemostat prevents the weak PUL extending and restores the contractility of the muscle forces (arrows). Cessation of urine loss on coughing confirms a loose PUL. This is the only method possible for clinically diagnosing a damaged pubourethral ligament (PUL). See also Videos 1 and 2.

The exact manoeuvre in Figure S6 was monitored with transperineal ultrasound to show changes in geometry and closure, Figure S7 and also, urodynamically, with pressure monitors in the bladder and urethra demonstrating pressure rise both at mid and distal urethra, Figure S8.

1.4 | Why laxity in the distal closure mechanism needs to be repaired

Laxity in the distal closure mechanism due to EUL laxity is a rarely recognized cause of non-stress urine leakage. The three anatomical structures which comprise the distal closure mechanism are the external urethral ligament, suburethral vagina (“hammock”) and the anterior portion of PCM, Figure S9. In the normal patient, the distal closure muscle stretches the distal vagina forwards between PUL and the external urethral meatus to close the distal urethra, (Figure S9). The classical symptoms of EUL defect are small leakages on walking, or moving suddenly, accompanied by a feeling like “a bubble of air escaping.” In patients who leak after MUS, EUL laxity (Figures S9 and S10), needs to be excluded.

1.5 | The rhabdospincter (RS) generates urethral pressure, but not closure.

The rhabdospincter, Figure S11, is a striated muscle with small nuclei which occupies the middle segment of the urethra. It is horseshoe shaped, sits outside the smooth muscle, inserts into it, with paucity, or absence of striated muscle fibres inferiorly. It becomes atrophied with age, often severely.⁷ Many consider RS to be the principal sphincter of the urethra. This was disproven in 1995.⁸ In an experiment carried out during a midurethral sling in 1995⁸ on four patients. Pressure measurements were made in exactly the same positions inside and outside the urethra with Gaeltec pressure transducers. With the vagina intact, with a measured 240 mL saline in the bladder, mean pressure during coughing at the high pressure zone was 47.6 cm H₂O (range 10-100 cm H₂O). Two of the four patients lost urine on coughing.

With open vaginal flaps, all patients lost large streams of urine on coughing, even though the intraurethral pressure was observed to rise between 78, 94, 112, and 170% during coughing. On tightening the flaps (without elevation) full continence was restored in all four patients. It was concluded from this that pressure increase arose from within the urethra, from the rhabdospincter, that the rhabdospincter was a weak muscle, incapable of closing the urethra and that continence was effected by the closure mechanisms described in 1990.¹ The conclusions that RS was a weak muscle incapable of sufficient urethral closure were similar to those of Huisman in 1983.⁸ Huisman noted 30% drop in MUP with bilateral pudendal block and urine loss with stress. However, there was a 500% increase in EMG activity with stress after pudendal block, which showed that the pudendal nerve did not supply the RS nor did RS contraction control continence (urethral closure). These experiments indicate that the role of the RS is not primarily to close the urethra, but to help seal it.

1.6 | What is the ultimate pathway to USI and ISD, pressure or resistance to flow?

In a urodynamically monitored pre and post-operative study,⁹ it was found that patients with ISD were cured with no increase in maximal urethral pressure post-operatively. With reference to Figure S11, as PUL lengthens from N to N + L, the area inside the urethra increases; the RS also lengthens, decreasing its contractile strength. Maximal urethral pressure (MUP) is measured by an intraurethral transducer. Pressure “P” = force/area (πr^2). Because force diminishes as urethral area increases, MUP will decrease: if it is below 20 cm H₂O, the patient will be diagnosed as having “intrinsic sphincter defect” (ISD). The closure mechanisms described in this work narrow the urethra. Narrowing causes an exponential rise in the intraurethral resistance, which varies inversely by the 4th power of the radius (Poiseuille's Law).¹⁰ Continence therefore revolves around the ability of the urethral

tube to have sufficient internal resistance to passage of urine from bladder to the outside. Resistance is consequential on the inverse of the 4th power of the radius, pressure only on the 2nd power, an insufficient indicator. This possibly explains why urethral pressure per se has been repeatedly shown to have no relationship to continence.¹¹

1.7 | Ligament damage is the primary cause of USI

Results from a blinded experiment¹² emphasized the importance of ligaments. A group of 47 women, mean age 46.8 years (range 18-78), had muscle biopsies of the PCM at the same time as a midurethral sling. Almost all biopsies showed evidence of severe muscle damage; nevertheless, 89% were cured the next day after a midurethral sling (MUS), suggesting that even severely damaged muscles seem to have sufficient contractile strength to restore urethral closure once the PUL has been restored.

1.8 | Pressure transmission theories and continence control

None of the experimental works described here can be explained by pressure transmission theories.^{13,14}

2 | CONCLUSIONS

The key element in restoration of urinary stress incontinence is creation of a competent PUL using the collagenous neoligament surgical principle described in Part 1. This creates a firm insertion point for the closure muscles PCM and LP, thereby restoring their contractile strength.¹⁵ Figure S6-8 and Videos 1&2 explain why in the original MUS,^{1,2} the tape was elevated until no urine leaked on coughing. At this critical tape length, the tape creates the artificial collagenous neoligament required for longer term cure.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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